

# Gout

## WHAT IS GOUT?

### The Processes in the Body Leading to Hyperuricemia and Gout

Gout is an arthritic condition (inflammation of the joints) that mostly affects men age 40 and older. It is nearly always associated with chronic *hyperuricemia*, a long-lasting abnormally high concentration of uric acid in the blood.

*Metabolism of Purines.* The process leading to hyperuricemia and gout begins with the metabolism of *purines*, which are nitrogen-containing compounds that are important for energy. Purines can be divided into two types:

- *Endogenous.* Endogenous purines are manufactured within human cells.
- *Exogenous.* Exogenous purines are obtained from foods.

All mammals *except* humans possess an enzyme called *uricase* that breaks purines down into a very soluble product called *allantoin*. Without uricase, purine ultimately breaks down into *uric acid*, which can build up in body tissues if it is not adequately eliminated in urine.

*Uric Acid and Hyperuricemia.* Uric acid is produced in the liver and enters the bloodstream. The path leading to high concentrations of uric acid and gout is the following:

- Most uric acid eventually passes through the kidneys and is excreted in the urine. The rest is disposed of in the intestines, where it is processed and broken down by bacteria.
- Normally these processes keep the concentration of uric acid in the blood plasma (the liquid part of the blood) at a healthy level, which is below 6.8 milligrams per deciliter (6.8 mg/dL).
- Under certain circumstances, however, the body produces too much uric acid or excretes too little. [ See What Causes Gout?] In either case, concentrations of uric acid increase in the blood. This condition is known as *hyperuricemia*.
- If concentrations of uric acid reach 7 mg/dL and above, the blood becomes supersaturated and needlelike crystals of a salt called *monosodium urate* (MSU) form.
- In time, as MSU crystals accumulate, they cause inflammation and pain, characteristic symptoms of gout.

### Gout and Other Conditions Associated with Hyperuricemia

High levels of uric acid are associated not only with gout but also with a number of other conditions. They can occur independently but may also develop one after the other if gout is untreated.

*Acute Gouty Arthritis.* Acute gouty arthritis is the stage at which the first symptoms of gout appear. It most often occurs in men.

*Chronic Tophaceous Gout and Tophi.* After several years, persistent gout develops called chronic tophaceous gout. This long-term condition often produces *tophi*, which are solid deposits of MSU crystals that form in the joints, cartilage, bones, and elsewhere in the body. In some cases, tophi break through the skin and appear as white or yellowish-white, chalky nodules that have been described as looking like crab eyes.

Without treatment, tophi develop on average about 10 years after the onset of the disease, although their first appearance can range from three to 42 years. They are more apt to appear early in the course of the disease in older people. In the elderly population, women appear to be at higher risk for tophi than men.

Today, drug therapy has reduced the prevalence of chronic tophaceous gout to as little as 3% of patients. Certain groups, such as transplant patients receiving cyclosporine, however, still face a high risk of developing tophi.

*Uric Acid Nephrolithiasis (Kidney Stones).* Uric acid nephrolithiasis occurs when kidney stones form from uric acid. In one study, however, patients with these stones were more likely to have elevated levels of uric acid in their blood than in their urine, suggesting that gout is responsible for these stones. Uric acid and other kidney stones are present in 10% to 25% of patients with primary gout, a prevalence more than 1,000 times that of the general population. In gout caused by other conditions (called secondary gout), the reported incidence reaches 42%.

It should be noted that uric acid stones can also form in the absence of gout or hyperuricemia. Also, not all of the kidney stones in patients with gout are composed of uric acid; some are composed of calcium oxalate, calcium phosphate, or those substances combined with uric acid. [ *See Kidney Stones.* ]

*Chronic Uric Acid Interstitial Nephropathy.* Chronic uric acid interstitial nephropathy occurs when crystals slowly form in the structures and tubes that carry fluid from the kidney. It is reversible and not likely to injure the kidneys.

*Kidney Failure.* Sudden overproduction of uric acid can occasionally block the kidneys and cause them to fail. This occurrence is very uncommon but can occur with the following conditions:

- After chemotherapy for leukemia or lymphoma.
- After severe heat stress from vigorous exercise.
- Following epileptic seizures.
- After corticosteroid therapy for severe allergic reactions.

## **WHAT CAUSES GOUT?**

Gout is classified as either primary (the most common type) or secondary, depending on the cause of the associated hyperuricemia (high levels of uric acid in the blood). In both types of gout, between 70% and 95% of hyperuricemia cases are the result of under-excretion of uric acid, rather than uric acid over-production.

Many people develop hyperuricemia, however, but not all people with the condition develop gout. Researchers are unable to determine the reason for this, or why gout develops in certain joints but not in others.

### **Primary Gout**

More than 99% of primary gout cases are referred to as idiopathic, meaning that the cause of the hyperuricemia cannot be determined. They are most likely due to a combination of hormonal and genetic factors that cause metabolic abnormalities resulting in overproduction of uric acid or reduced excretion of uric acid. The remaining 1% of primary gout cases are traceable to either of two rare inherited enzyme defects that affect purine synthesis in the cells.

### **Secondary Gout**

In secondary gout, hyperuricemia is caused by drug therapy or by medical conditions other than an inborn metabolic disorder that increase uric acid concentration.

*Alcohol Use.* Alcohol use is a major contributor to gout and increases uric acid levels in three ways:

- By providing an additional dietary source of purines (the compounds from which uric acid is formed).
- By intensifying the body's production of uric acid.
- By interfering with the kidneys' ability to excrete uric acid.

*Renal (Kidney) Insufficiency and Its Causes.* Hyperuricemia occurs in between 30% and 85% of people who have renal (meaning kidney) insufficiency. Renal insufficiency is a major cause of gout in older people. This results in an impaired ability of the kidneys to eliminate waste products, including uric acid, which then build up in the blood. This condition, in turn, can be the result of the following:

- Thiazide diuretics (the "water pills" used to control hypertension). These agents are very highly associated with gout. In fact, 75% of elderly-onset gout patients report the use of diuretics.
- Organ transplantation . Kidney transplantation poses a high risk for renal insufficiency and gout. In addition, other transplantation procedures, such as heart and liver, increase the risk. The procedure itself poses a risk. In addition, cyclosporine an immunosuppressive agents used after these procedures to help prevent rejection of the implant, poses a particular risk for gout. (Alternative agents may be effective and pose less of a risk.)

*Other Medications.* The list of drugs that cause hyperuricemia is long. In addition to diuretics and immunosuppressants, other agents that increase the risk for gout include the following:

- Pyrazinamide (used to treat tuberculosis).
- *Low* doses (not high doses) of aspirin reduce uric acid excretion and increase the chance for hyperuricemia. This may be a problem for older people who take baby aspirin (80 mg) to protect against heart disease. (High doses have the opposite effect.)
- Niacin.

*Other Conditions.* A number of other conditions can cause gout. They include the following:

- Leukemia.
- Lymphoma.
- Psoriasis.
- Over exposure to lead.

## **WHAT ARE THE SYMPTOMS OF GOUT?**

Gout is often divided into four symptomatic stages:

- Asymptomatic hyperuricemia.
- Acute gouty arthritis.
- Intercritical gout.
- Chronic tophaceous gout.

These stages may differ depending on the age of onset:

- In middle-aged adults, symptoms are more likely to occur in one joint, most often in the lower limbs. About 60% of cases in this age group first occur in the big toe.
- In elderly people, symptoms are more likely to occur in a number of joints in the upper extremities, particularly the fingers.

### **Asymptomatic Hyperuricemia**

Asymptomatic hyperuricemia, in which MSU slowly builds up, always precedes gout and is considered the first stage of the disorder. It lasts for an average of 30 years.

Note: Hyperuricemia does not inevitably lead to gout. In fact, less than 20% of the hyperuricemic population develops the full-blown arthritic disease.

### **Acute Gouty Arthritis**

Acute gouty arthritis occurs when the first symptoms of gout appear. Sometimes gout is heralded by brief twinges of pain (petit attacks) in an affected joint, which can precede the actual full-blown condition by several years. MSU crystals form at normal body temperature when concentrations in the blood reach 7 mg/dL. At lower temperatures, crystals form at lower concentrations. Since blood temperature falls with distance from the heart, gout strikes the toes and fingers first.

The symptoms of acute gout arthritis are described as follows:

- The primary symptom is severe pain at and around the joint. Some patients describe it "crushing" or resembling a dislocated bone. The area can be so tender that walking and even the weight of bed sheets can be unbearable. One writer described gout in the toe as "walking on my eyeballs." The pain usually takes eight to 12 hours to develop. In many cases the attack occurs late at night or early in the morning and announces itself by waking the sufferer.
- Swelling may extend beyond the joint, indicating fluid build-up within.
- The skin over the affected area is often red, shiny, and tense. After a few days it may start to peel.
- Chills and mild fever, loss of appetite, and feelings of ill health may occur with an attack.

Most often symptoms first start in one joint, a condition is called *monoarticular gout*. If more than one joint is affected, it is known as *polyarticular gout*. (Multiple joints are affected in only 10% to 20% of first attacks.)

- *Monoarticular Gout*. The joint of the big toe is the site of about 60% of all first monoarticular gout attacks in middle-aged adults. This occurrence is known as *podagra*. (The site is medically referred to as the big toe's metatarsophalangeal joint, the point where one of the long five bones of the foot meets the first digit of a toe.) Symptoms can also occur in other locations, although most often they develop somewhere on one lower limb in middle-aged men.
- *Polyarticular Gout*. Older people are more likely to have polyarticular gout. In this condition, the joints of the foot, ankle, knee, wrist, elbow, and hand are the most frequently affected. The pain usually occurs in joints on one side of the body and it is usually, although not always, in the lower extremities. People with polyarticular gout are more likely to have a more gradual onset of pain and a longer delay between attacks. Older people are at higher risk for polyarticular gout than younger adults and it tends to occur in the upper extremities, often in the fingers. People with polyarticular gout are also more likely to experience the low-grade fever, loss of appetite, and a general feeling of poor health.

An untreated attack will typically peak 24 to 48 hours after the initial appearance of symptoms, and subside after five to seven days, although it can last only hours to as long as several weeks.

### **Intercritical Gout**

Intercritical gout is the term used to describe the periods between attacks. The first attack is usually followed by a complete remission of symptoms, but left untreated, gout nearly always recurs at some point in the future. One study reported that 62% of subjects

experienced at least one further attack within a year. At the end of two years, 78% of patients experienced a recurrence. After 10 years, 93% of the patients had had repeat attacks.

### **Symptoms of Chronic Tophaceous Gout**

*Development of Chronic Pain.* When gout remains untreated, the intercritical periods typically become shorter and shorter, and the attacks, although sometimes less intense, can last longer. Over the long term (about 10 to 20 years) gout becomes a chronic disorder characterized by constant low-grade pain and mild or acute inflammation. Gout may eventually affect several joints, including those that may have been free of symptoms at the first appearance of the disorder. In rare cases, the shoulders, hips, or spine are affected.

*Symptoms of Tophi.* Tophi, the knobby MSU crystal deposits that form during chronic gout, generally form in the following location:

- Helix of the outer ear (the curved ridge along the edge of the ear).
- Forearms.
- Elbow or knee.
- Hands or feet. (Older patients, particularly women, are more likely to have gout in the small joints of the fingers.)
- In rare cases, they can settle in regions around the heart and spine.

Tophi, generally, are painless. However, they can often cause pain and stiffness in the affected joint. Eventually, they can also erode cartilage and bone, ultimately destroying the joint. Large tophi under the skin of the hands and feet can give rise to extreme deformities.

### **Triggers for Gout Symptoms**

Gout symptoms may be precipitated by various conditions including the following:

- Severe illness (an important trigger). Between 20% to 86% of patients with gout experience a recurrence when they are hospitalized. Gout accompanies and can be exacerbated by serious conditions that are associated with kidney and heart disease including diabetes, obesity, unhealthy cholesterol levels, insulin resistance, and high blood pressure.
- Stress.
- Infection.
- Joint injury.
- Weight loss.
- Surgery.

- Certain drug treatment (an important trigger).
- Overindulgence in alcohol or purine-rich foods.
- Over-strenuous exercise. Even a long walk can trigger symptoms in a patient who is not sufficiently physically fit.

Symptoms occur more frequently in the spring, with the peak in April, according to some studies.

## **HOW SERIOUS IS GOUT?**

Gout rarely poses a long-term health threat if properly treated. It does, however, remain a source of short-term pain and incapacity for thousands of Americans.

### **Pain and Disability**

Left untreated, gout can develop into a painful and disabling chronic disorder. Persistent gout can destroy cartilage and bone, causing irreversible joint deformities and loss of motion. Tophi can grow to the size of handballs and can destroy bone and cartilage in the joints, similar to the process in rheumatoid arthritis. If they lodge in the spine, tophi can cause serious damage including compression, although this is very rare. In extreme cases, joint destruction results in complete disability.

### **Kidney Conditions**

*Kidney Stones.* Kidney stones occur in between 10% and 40% of gout patients, and can occur at any time after the development of hyperuricemia. Although the stones are usually composed of uric acid, they may also be mixed with other materials.

*Kidney Disease.* About 25% of patients with chronic hyperuricemia develop progressive kidney disease, which sometimes ends in kidney failure. It should be noted, however, that many experts believe that chronic hyperuricemia is unlikely to be a common cause of kidney disease. In most cases, the kidney disease comes first and causes high concentrations of uric acid.

### **Gout and Heart Disease**

Gout often accompanies heart problems, including high blood pressure, coronary artery disease, and congestive heart failure. Hyperuricemia, in fact, has been associated with a higher risk of death from these conditions. One 2001 study reported that disease activity in gout may contribute to unhealthy cholesterol and lipid levels. Some interesting evidence, however, suggests that hyperuricemia may occur as a response to inflammatory damage that occur with heart disease and may even be protective.

### **Other Medical Conditions Associated with Gout**

The following are some conditions that are associated with long-term gout:

- Cataracts.
- Dry eye syndrome.
- Complications in the lungs (in rare cases, uric acid crystals occur in the lungs).

## WHAT ARE THE RISK FACTORS FOR GOUT?

Risk factors are attributes or activities associated with a greater-than-normal likelihood of developing a particular disorder. Sometimes a causal connection between the attribute or activity and the disorder can be established, but at other times there is simply a statistical correlation. The risk factors for gout, of which there are several, are identical to those for hyperuricemia.

### Prevalence

Gout is one of the most common types of arthritis. Based on self-reports, gout is estimated to affect about 2.1 million Americans (1.56 million men and 550,000 women). Some experts believe, however, that this may be an overestimate. The prevalence of gout has been rising in recent decades, not only in America but in other developed countries, possibly because of dietary and lifestyle changes, greater use of medications that cause hyperuricemia, and aging populations.

Gout is very uncommon in less-developed countries, however, and in 1952 it was said to be unknown in China, Japan, and the tropics.

### Age

*Middle-Aged Adults.* Gout usually first occurs in middle-aged men, and peaks in mid-40s. It is most often associated in this age group with obesity, high blood pressure, unhealthy cholesterol levels, and heavy alcohol use.

*Elderly.* Gout can also first develop in older people, when it occurs equally in men and women. In this group, gout is most often associated with kidney problems and the use of diuretics. It is less often associated with alcohol use.

*Children.* Among children, the levels of uric acid in both girls and boys are low, on average 3 to 4 mg/dL. Except for rare inherited genetic disorders that cause hyperuricemia, gout in children is almost unheard of.

### Gender

*Men.* Men are significantly at higher risk for gout. In males, uric acid levels rise substantially at puberty, with the result that the level exceeds 7 mg/dL (considered to indicate hyperuricemia) in about 5% to 8% of American men. Gout typically strikes only after 20 to 40 years of persistent hyperuricemia, however, so men who develop it usually experience their first attack between the ages of 30 and 50 years. In one study that

followed male medical students for 28 years, the prevalence of gout was 5.8% in Caucasian men and 10.9% in African American men.

*Women.* Before menopause, women have a significantly lower risk for gout than men, possibly because of the actions of estrogen. This female hormone appears to facilitate uric acid excretion by the kidneys. (Only about 15% of female gout cases occur before menopause.) After menopause the risk increases in women so that after age 60 the incidence is equal in men and women, and after 80, gout occurs actually more often in women.

### **Family History**

A fairly substantial proportion of patients with gout (10% to 20%) has a family history of gout. According to a 2001 Taiwan study, patients with possible inherited conditions were more likely to have an earlier onset (about 41 years) compared to those whose gout is due to other factors (48 years). They were also more likely to have family histories of obesity, type 2 diabetes, and kidney insufficiencies.

### **Other Risk Factors**

*Obesity.* Researchers report a clear link between body weight and uric acid levels. In one Japanese study, overweight people had between two and over three times the incidence of hyperuricemia as those of normal or low weights. Obesity may be an especially important risk factor for gout in men. Children who are obese may have a higher risk for gout in adulthood.

*Hypertension and Diuretics.* The use of diuretics, which are agents used to treat high blood pressure, are highly associated with gout. Hypertension (high blood pressure), itself, is found in 25% to 50% of patients with gout, but whether it causes hyperuricemia is uncertain.

*Alcohol Use.* Alcohol use is highly associated with gout in younger adults. Binge drinking particularly increases uric acid levels. It appears to play less of a role among elderly patients, especially among women with gout.

*Thyroid Dysfunction.* Some studies have reported a higher prevalence of gout in people with hypothyroidism (low levels of thyroid hormone). There is also some evidence to suggest that *hyper*thyroidism (high levels of thyroid hormone) can increase uric acid levels, although not to the degree that low thyroid hormones levels do.

## **HOW IS GOUT DIAGNOSED?**

### **Medical History and Physical Examination**

Determining which joints are affected is an obvious first step in any diagnosis. A physical examination and medical history can reveal a number of significant indicators that help confirm or rule out gout. The following are some examples:

- Gout is more likely if arthritis first appears in the big toe than if it first appears elsewhere.
- The speed of the onset of pain and swelling is relevant; symptoms that take days or weeks rather than hours to develop probably indicate a disorder other than gout.
- Abnormal enlargements in joints that had been affected by previous injury or osteoarthritis are possible signs of gout. This is particularly significant in older women on diuretics.

### **Examination of Synovial Fluid**

Examination of synovial fluid is the most accurate method for diagnosing gout. It may even be helpful in detecting gout during intercritical periods. The synovial fluid is the lubricating liquid that fills the *synovium* (the membrane that surrounds a joint and creates a protective sac). The fluid cushions joints and supplies nutrients and oxygen to cartilage, the slippery tissue that coats the ends of bones.

*Procedure.* The procedure for taking a sample of synovial fluid from an affected joint is called aspiration:

- A needle attached to a syringe is inserted into the joint and suction is used to draw the fluid into the syringe.
- Local anesthesia is avoided because it can reduce the effectiveness of aspiration, but normally the procedure is only mildly uncomfortable.
- Following the procedure there can be some minor discomfort in the area where the needle was inserted, but it usually dissipates quickly.

Aspiration can cause infection, though this occurs in less than 0.1% of patients. Aspiration sometimes eases a patient's symptoms by reducing swelling and pressure on the tissue surrounding the joint.

*Analyzing the Fluid.* After the sample is taken, it is sent to a laboratory, where a specialist examines the sample through a microscope under polarized light. This special light will reveal the presence of monosodium urate (MSU) crystals, which will nearly always confirm a diagnosis of gout. The laboratory can also test the sample for infection.

### **Blood Test for Uric Acid Levels**

A blood test is usually given for measuring uric acid and detecting hyperuricemia. A low level of uric acid in the blood makes a diagnosis of gout much less probable, and a very high level increases the likelihood of gout. Some experts argue, however, that such measurements are not very useful, given what is known about the variability of uric acid levels in people with gout:

- Uric acid levels in the blood during an attack of gout can lie within or below the normal range.
- Even if hyperuricemia is present, it is very common in the population and does not necessarily indicate the presence of gout.

## Determining Uric Acid Excretion in Urine

It is sometimes helpful to gauge the amount of uric acid excreted by the patient, particularly if the patient is young and has pronounced hyperuricemia that might be related to a metabolic disorder. If uric acid exceeds a particular value in the urine, further tests for an enzyme defect or other identifiable cause of gout arising from uric acid overproduction are justified. Greater-than-normal amounts of uric acid in the urine also indicate that the patient faces a greater risk of developing uric acid kidney stones, and can guide the physician in his or her choice of drug therapy for chronic gout.

*24-Hour Urine Sample.* Typically, urine samples are taken over the course of 24 hours. To provide a urine sample, the following steps are taken:

- The urine is collected during an intercritical period, after the patient has been placed on a purine-reduced diet. The patient is also asked to temporarily stop using alcohol and any medications that can interfere with the test.
- The patient should not change any of his or her usual eating or drinking patterns when performing this test.
- The patient discards the first urination on the day of the test.
- Afterward all urine passed over the next 24 hours is collected, including the first urination on the morning of day two.
- The container is then delivered to the patient's physician or sent directly to the laboratory.

## Imaging Techniques

*X-Rays.* For the most part, x-rays do not reveal any abnormalities during the early stages of gout, and their usefulness where gout is concerned lies in assessing the progress of the disorder in its chronic phase and in identifying other health problems whose symptoms may resemble those of gout. Tophi can be seen on x-rays before they become apparent on physical examination.

*Advanced Imaging Techniques.* Advanced imaging techniques being investigated for identifying tophi include computed tomography (CT), magnetic resonance imaging (MRI), and Doppler ultrasonography (US). A 2002 study comparing these approaches found that CT scans offered the best images.

## Ruling Out Other Disorders

As part of the diagnosis, other disorders that produce gout-like symptoms or cause hyperuricemia should be ruled out. In general, it is easy to distinguish acute gout that occurs in one joint from other arthritic conditions. The two disorders that may confuse this diagnosis are pseudogout and septic arthritis. Chronic gout can often resemble rheumatoid arthritis. A number of other conditions may at some point in their course resemble gout. [ *See Conditions with Similar Symptoms to Gout.* ]

*Pseudogout.* Pseudogout is a condition most likely to be confused with gout. [For details on this condition, *See Box Pseudogout.*]

#### **PSEUDOGOUT (CALCIUM PYROPHOSPHATE DIHYDRATE DEPOSITION DISEASE)**

##### **What Is Pseudogout and How Is It Different from Gout?**

Pseudogout, is very similar to gout, but is caused by deposits of calcium pyrophosphate dihydrate crystals in and around the joints. (It is, in fact, medically referred to as calcium pyrophosphate dihydrate deposition disease, or CPPD.) Though pseudogout resembles gout in some ways, there are differences:

- The first attack typically strikes the knee rather than the joint of the big toe but it may involve any joint. At least two-thirds of cases affects more than one joint during a first attack.
- The symptoms of pseudogout also appear more slowly than those of gout, taking days rather than hours to develop.
- Pseudogout is more likely to first develop in elderly people, particularly those with osteoarthritis. (It affects between 10% and 15% of people over 65.)
- Pseudogout is more likely to occur in the autumn while gout attacks are most common in the spring.

##### **How Serious is Pseudogout?**

There is no cure for pseudogout. It is a progressive disorder that can eventually destroy joints.

##### **How is Pseudogout Treated?**

Treatments for pseudogout are similar to those for gout and are aimed at relieving the pain and inflammation and reducing the frequency of attacks:

- NSAIDs are effective for treating inflammation and pain from pseudogout.
- Oral magnesium carbonate may help dissolve crystals but existing hard deposits remain.
- For acute attacks in large joints, fluid aspiration alone or with corticosteroids may help.
- Intravenous colchicine is also helpful but may be toxic.
- Surgery may be required for joint replacement.

*Rheumatoid Arthritis.* Rheumatoid arthritis can cause distortion in the joints of the fingers, inflammation, and pain that may mimic gout. It is particularly difficult to distinguish chronic gout in older people from rheumatoid arthritis. A proper diagnosis can be made with a detailed medical history, laboratory tests, and identification of MSU crystals.

*Osteoarthritis.* Gout can coincide and be confused with osteoarthritis in older people, particularly when it occurs in arthritic finger joints in women. In general, gout should be

suspected if the joints in the fingers tips are unusually enlarged.

*Infections.* Joint infections can have features that resemble gout and a correct diagnosis is critical for appropriate treatment. For example, some cases of gout have been confused with infection after joint replacement. On the other hand, joint infection not associated with surgery might indicate sepsis, which is a widespread and potentially life-threatening bacterial infection that can cause inflamed joints, chills, and spiking fever. The severity of the fever and a high white-blood cell count in the joint fluid helps diagnose a septic infection, while identifying urate crystals in the joint is a good indicator of gout.

*Charcot Foot.* Between 1% and 2.5% of people with diabetes suffer from Charcot foot or Charcot joint (medically referred to as neuropathic arthropathy). This condition is caused by abnormalities in the nerves in the feet. Early changes may resemble gout, with the foot becoming swollen, red, and warm. Recognition and treatment of this condition is very important. A seriously affected foot can become deformed. The bones may crack, splinter, and erode, and the joints may shift, change shape, and become unstable.

*Bunions.* A bunion is a deformity that usually occurs at the head of the first of five long bones (the metatarsal bones) that extend from the arch and connect to the toes and may be confused with gout. The first metatarsal bone is the one that attaches to the big toe. A bunion begins to form when the big toe is forced in toward the rest of the toes, causing the head of the first metatarsal bone to jut out and rub against the side of the shoe; the underlying tissue becomes inflamed, and a painful bump forms. As this bony growth develops, the bunion is formed as the big toe is forced to grow at an increasing angle towards the rest of the toes.

<b>Some Diseases with Similar Symptoms to Gout</b>	
<b>Disease</b>	<b>Specific Subtypes</b>
Osteoarthritis	
Infectious Arthritis	Lyme disease, septic arthritis, bacterial endocarditis, mycobacterial and fungal arthritis, viral arthritis, osteomyelitis
Postinfectious or Reactive Arthritis	Reiters syndrome (a disorder characterized by arthritis and inflammation in the eye and urinary tract), rheumatic fever, inflammatory bowel disease
Pseudogout	
Rheumatic Autoimmune Diseases	Rheumatoid arthritis, systemic vasculitis, systemic lupus erythematosus, scleroderma, Still's Disease (also called juvenile rheumatoid arthritis)
Fibromyalgia	
Other Diseases	Chronic fatigue syndrome, hepatitis C, familial Mediterranean fever, cancers, AIDS, leukemia, bunions, Whipple's disease, dermatomyositis, Behcet's

disease, Henoch-Schonlein purpura, Kawasaki's disease, erythema nodosum, erythema multiforme, pyoderma gangrenosum, pustular psoriasis
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## WHAT ARE THE GENERAL GUIDELINES FOR TREATING GOUT?

Acute attacks of gout and long-term treatment of gout and its associated hyperuricemia require different approaches. All phases are treated mainly with drugs. There are also specific treatment regimes for conditions associated with gout, including uric acid nephropathy and uric acid nephrolithiasis.

### Lifestyle Measures

Many patients do not require medications. During the period between gout attacks, patients are advised to avoid foods high in purines and to maintain a healthy weight. Patients should also avoid alcohol and reduce any stress. [ *See What Lifestyle Measures Can Help Prevent Gout?*]

### Treatments for Asymptomatic Hyperuricemia

Because asymptomatic hyperuricemia usually does not lead to gout or other health problems, and would have to be treated with drugs that present certain risks and can be expensive, treatment to prevent a first attack of gout in hyperuricemic patients is considered inadvisable. In unusual circumstances, for example when very high uric acid levels threaten the kidney, treatment may be justified.

### Treatment of an Acute Attack of Gout

Drug treatments for acute attacks of gout are aimed at relieving pain and reducing inflammation. They should be started as early as possible.

- NSAIDs. Powerful forms of nonsteroidal anti-inflammatory drugs (NSAIDs) are the drugs of choice for an acute attack in younger, healthy patients with no serious health problems.
- Corticosteroids or corticotropin (ACTH). Corticosteroids or corticotropin (ACTH) may be used in patients who cannot tolerate NSAIDs and they may be particularly beneficial for elderly patients. Injections into the affected joints provide effective relief for many patients. Oral steroids may be used for patients who cannot take NSAIDs or colchicine.
- Colchicine. Colchicine is used in healthy adults only and with caution. It should not be used in patients with kidney or liver problems or in pregnant women.

Rest and protecting the affected joint with a splint can also promote recovery. A 2002 study reported that applying ice packs for 30 minutes four times daily significantly reduced pain. Interestingly, one 2001 study recommended applying *warm* water continuously and moving the joint. The theory behind this advice was that the pain in a gout attack is due to grinding from the crystals and that warmth would help dissolve the crystals and relieve pain.

After the first attack, some physicians advise their patients to keep a supply of medications on hand so that self-medication can begin at the first sign of symptoms of a second acute attack.

### **Treatments to Prevent Attacks during Intercritical Gout**

After an acute attack patients remain at risk for another for several weeks during the intercritical period. In such cases, low doses of either of the following agents may be used to during this period for prevention.

- Colchicine.
- NSAIDs.

These agents should be taken in low doses for one to two months after an attack or longer in patients who have experienced frequent attacks. These are simply anti-inflammatory drugs, however, and have no effect on hyperuricemia. [For Description of individual drugs see What Are the Specific Drugs Used for Gout? *below*.]

### **Drugs Used to Reduce Uric Acid Levels in Chronic Gout**

In some cases, patients will use agents (antihyperuricemic drugs) to reduce uric acid levels. The goals of antihyperuricemic therapy are to reduce the frequency of attacks and to dissolve monosodium urate (MSU) crystals and tophi. In fact, a 2001 study suggested that patients with chronic gout must maintain uric acid levels at or below 6 mg/dL in order to prevent further attacks.

*Candidates.* Long-term treatment of hyperuricemia may be recommended for the following situations:

- There is a risk for tophaceous gout.
- The patient has suffered more than two or three acute attacks of gout.
- Attacks are unusually severe or affect more than one joint.
- X-rays show joint damage from gout.
- Hyperuricemia is caused by an identifiable inborn metabolic deficiency.

Normal kidney function is essential for taking these drugs. This therapy, then, may not be as beneficial in many elderly patients, who often have some kidney insufficiency.

*Agents Used to Reduce Uric Acid.* A number of effective antihyperuricemic agents are available. In general, their effects differ depending on whether a patient's high uric acid is due to overproduction or a failure to eliminate enough in the urine. [For Description of individual drugs see What Are the Specific Drugs Used for Gout? *below*.] They including the following:

- Allopurinol. Allopurinol inhibits uric acid production and is useful for those who overproduce uric acid, who have kidney disorders, or who have kidney stones.

- Uricosurics. (Most often probenecid and sulfinpyrazone.) They are appropriate when gout is caused by under-excretion of uric acid, which occurs in about 80% of cases. They are not used for patients with reduced kidney function or those with tophaceous gout.

Certain steps must be made in undertaking hyperuricemic therapy:

- Some experts recommend a 24-hour collection sample in patients with frequent gout attacks to determine whether they are over-producers or under-excretors of uric acid.
- Before starting one of these drugs, any previous acute attack should be completely controlled and the joints should not be inflamed. Some physicians prefer to wait about a month after an attack.
- Low doses of NSAIDs or colchicine are used during several months after introducing anti-hyperuricemic therapies to prevent gout attacks that can occur. It should be noted that NSAIDs, particularly aspirin, as well as other salicylate drugs, interfere with uricosuric drugs and reduce effectiveness, so they should be avoided if possible by patients taking these agents.

The decision to use anti-hyperuricemic and if so, at what point, is not entirely clear, however. Some physicians do not prescribe them if hyperuricemia is mild or until a patient has had two attacks. Others prescribe them immediately after a single attack. Most of the time, antihyperuricemic therapy means taking a drug routinely throughout life, which many people find difficult to adhere to.

#### **Warning Note on Drug Treatments for Gout**

It should be noted that many drugs used for gout can also *precipitate* acute gout symptoms and so should not be used until symptoms have subsided. The patient should then start with small doses that gradually increase.

#### **Surgery**

Surgery is sometimes used to remove large tophi that are draining, infected, or interfering with the movement of joints. Several other surgical procedures are available for relieving pain in and improving the function of affected joints. It is sometimes necessary to replace joints.

## **WHAT ARE THE SPECIFIC DRUGS USED FOR GOUT?**

### **Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)**

Nonsteroidal anti-inflammatory drugs (NSAIDs) block prostaglandins, the substances that dilate blood vessels and cause inflammation and pain. They are the drugs of choice for young, healthy adults without any other serious medical condition. NSAIDs are usually taken orally at their highest safe dosage as long as gout symptoms persist and for three or four days after. Low doses of NSAIDs may be used to prevent gout attacks, including in patients who are starting anti-hyperuricemic therapies.

*NSAIDs Used.* Indomethacin (Indocin) in doses up to 200 mg a day is the usual choice for

gout. The first dose of indomethacin usually begins to act against the pain and inflammation within 24 hours and often much sooner. Ibuprofen, naproxen, sulindac, or others are good alternatives particularly for elderly patients who might experience confusion or bizarre sensations with indomethacin. Aspirin is an NSAID, but is associated with a *higher* risk for gout and should be avoided. There are dozens of NSAIDs available. [See Box Ulcers and Gastrointestinal Bleeding, for a list of NSAIDs].

*Side Effects and Complications.* Regular use of even over-the-counter NSAIDs may be hazardous for anyone and has been associated with the following side effects:

- Ulcers and gastrointestinal bleeding. This is the major danger with long-term use of NSAIDs. No NSAIDs, even over-the-counter brands, should be used for long-term pain relief except under physician direction. For example, an analysis of controlled trials reported that about 1% of patients taking aspirin over a 28-month period will experience gastrointestinal bleeding. A significant risk existed even at low doses or with the use of modified-release formulations. [ See Box Ulcer Risk by Specific NSAIDs.]
- Increased blood pressure. This is a particular problem in those on medications to reduce hypertension. Piroxicam (Feldene), naproxen (Aleve), and indomethacin (Indocin) appear to pose the greatest risks for high blood pressure. (Sulindac has the smallest effect.) People with hypertension, severe vascular disease, kidney, or liver problems, and those taking diuretics must be closely monitored if they need to take NSAIDs.
- Dizziness, ringing in the ear.
- Headache.
- Skin rash.
- Depression has also been noted.
- Confusion or bizarre sensation (in some higher-potency NSAIDs, such as indomethacin).
- NSAIDs may pose a higher risk for kidney injury, which would be of concern in patients with kidney problems, which might affect a number of gout patients. Any sudden weight gain or swelling should be reported to a physician.
- Diabetics taking oral hypoglycemics may need to adjust the dosage if they also need to take NSAIDs because of possible harmful interactions between the drugs.

<b>Ulcer Risk by Specific NSAIDs</b>		
<b>Lowest Risk</b>	<b>Medium Risk (see note)</b>	<b>Highest Risk</b>
Nabumetone (Relafen)	Aspirin	Flurbiprofen (Ansaid)
Etodolac (Lodine)	Ibuprofen (Motrin, Advil, Nuprin, Rufen)	Piroxicam (Feldene)
Salsalate	Naproxen (Aleve, Naprosyn, Naprelan, Anaprox)	Fenoprofen
Sulindac	Diclofenac (Voltaren)	Indomethacin (Indocin)
		Meclofenamate (Meclomen)

(Clinoril)	Tolmetin (Tolectin)  NOTE: Drugs within the medium risk group vary in risk. For example, studies show that use of naproxen is twice as likely as ibuprofen to be associated with hospitalization from GI bleeding.	Oxaprozin  Ketoprofen (Actron, Orudis KT)  NOTE: Ketoprofen is often considered a medium-risk drug, but one study reported that taking the drug even one week at low doses causes significant GI injury.
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*Drugs for Prevention of NSAID-Induced Ulcers.* For people who need to take NSAIDs regularly, some agents are available that may protect against bleeding and ulcers.

- Proton-pump inhibitors include omeprazole (Prilosec), lansoprazole (Prevacid), rabeprazole (Aciphex), and pantoprazole. Proton pump inhibitors are possibly the most protective agents and can actually heal existing ulcers. Their use has been demonstrated to reduce NSAID-ulcer rates by as much as 80% compared with no treatment.
- *Misoprostol.* Misoprostol is a prostaglandin, the protective substance blocked by NSAID use. It protects against the major intestinal toxicity of NSAIDs. It is used to prevent NSAID-induced ulcers, both duodenal and gastric, but is not useful in healing existing ulcers.
- *H2 Blockers.* Some over-the-counter H2 blockers, particularly famotidine (Pepcid AC) or ranitidine (Zantac) may help prevent NSAID-induced ulcers. [For prevention of NSAID-caused ulcers see *Peptic Ulcers.*]

### **COX-2 Inhibitors**

Celecoxib (Celebrex), rofecoxib (Vioxx), and valdecoxib (Bextra) are known as COX-2 (cyclooxygenase-2) inhibitors. They may prove to be beneficial for chronic tension-type headache without incurring as high risk for ulcers and bleeding. Meloxicam (Mobic) is a new drug known as a COX-2 preferential; ie, it inhibits COX-2 more than COX-1. It is not known yet if its actions differ significantly from standard NSAIDs.

*Benefits.* These agents may prove to be as effective and less harmful to the GI tract than NSAIDs. Importantly, studies are reporting a lower incidence of ulcers and other toxic side effects in patients taking the COX-2 inhibitors than in those taking NSAIDs. The drugs were all equally effective in relieving pain. (One study compared celecoxib with the NSAIDs ibuprofen or diclofenac and the other compared rofecoxib with the NSAID naproxen.) One 1999 study even found the rate of GI problems with celecoxib was equal to that in people who do not take NSAIDs at all. COX-2 inhibitors are currently more expensive than traditional NSAIDs, however, and some insurers do not pay for them.

Theoretically, they may even have properties that produce less adverse effects on cartilage than NSAIDs may have.

Some early evidence also suggests they may be protective against colon cancer and possibly even Alzheimer's disease.

*Possible Negative Effects.* In spite of their promise, some researchers theorize that inhibiting COX-2 may have some negative side effects over the long term:

- Although COX-2 inhibitors are very likely to have a lower risk for ulcers and GI bleeding than standard NSAIDs, studies have been mixed on whether patients taking COX-2 inhibitors have the same gastrointestinal symptoms (eg, diarrhea, abdominal discomfort) as standard NSAIDs. Vioxx may pose a higher risk for symptoms than Celebrex. (Other side effects found with short-term use include headache, and dizziness.)
- COX-2 inhibitors may have adverse effects on kidney function, particularly in elderly people, that were similar to the effects of standard NSAIDs. This effect can also trigger fluid build up and high blood pressure. (Celebrex may have fewer of these effects than Vioxx.)
- Patients taking anticoagulant drugs may experience a higher risk for bleeding with the use of these agents.
- Studies are reporting a higher incidence of heart attacks in patients taking Vioxx and possibly Celebrex than in those taking a standard NSAID, naproxen. Evidence suggests that both COX-2 inhibitors may increase the risk for blood clots. Experts also suggest that heart patients with chronic pain may be substituting COX-2 inhibitors for heart-protective NSAIDs (such as aspirin, ibuprofen, or possibly naproxen). Patients with heart disease who are taking low-dose aspirin should continue it even while they are taking COX-2 inhibitors.
- There have been reports of psychiatric side effects (hallucinations), liver toxicity, fluid build up, high blood pressure, and excess potassium in the blood with celecoxib or rofecoxib, usually with high doses.
- They may have negative effects on pregnancy and fertility.
- No one who has allergic reactions, hives, or asthma from sulfa drugs, aspirin, or other NSAIDs, should take a COX-2 inhibitor.
- The use of COX-2 inhibitors can interfere with many other drugs taken concurrently, including many taken for heart disease and high blood pressure. Patients should discuss all other medications with their physician.

More research is needed to confirm or refute any possible hazard.

#### **Other Investigative Alternatives to NSAIDs**

*NO-NSAIDs.* Experimental agents are being developed that combine nitric oxide with NSAIDs (NO-NSAIDs). Nitric oxide increases blood flow in the mucous lining and secretions of mucus and bicarbonate. Combining nitric oxide with NSAIDs may provide benefits similar to the COX-2 inhibitors.

*Arthrotec.* Arthrotec is a combination of misoprostol and the NSAID diclofenac that may

reduce the risk for gastrointestinal bleeding. One study found that patients taking Arthrotec had 65% to 80% fewer ulcers than those who took NSAIDs alone.

### **Colchicine**

Colchicine, a derivative of the autumn crocus (also called the meadow saffron), has been used against gout attacks for centuries. It is highly effective though no longer the first drug of choice because of its frequent, unpleasant, and sometimes very serious side effects.

*Oral Regimen.* The oral regimen requires doses every hour until the symptoms either improve or side effects develop; improvement should be evident by the tenth dose. Oral colchicine usually eliminates the pain of an acute attack within 48 hours.

The drug is generally not appropriate for elderly patients or those with kidney, liver, or bone marrow disorders. It can also effect fertility and should not be used during pregnancy.

Colchicine is unsuitable for many other patients as well, however, because of gastrointestinal side effects, which occur at the high doses necessary to relieve symptoms. They include nausea, vomiting, diarrhea, or abdominal cramps. *Note:* The antibiotic erythromycin or H2 blockers, such as famotidine (Pepcid AC), cimetidine (Tagamet), ranitidine (Zantac), may intensify the gastrointestinal side effects of colchicine.

Low doses do not pose as high a risk for gastrointestinal symptoms, and can prevent further attacks, including in patients who are starting anti-hyperuricemic therapies. Taking low doses has virtually no GI side effects.

*Intravenous.* Intravenous administration of colchicine relieves episodes of gout without gastrointestinal effects and for a time, physicians hoped it could be used routinely. The intravenous route has some serious side effects, however, and poses an increased risk for injury to the kidney, liver, central nervous system, and bone marrow.

*Warning Note:* Overdose of colchicine can be fatal, and there have even been reports of fatalities. The agent may also suppress blood cell production and cause nerve and muscular injury in certain people, sometimes even in those not taking high doses.

### **Corticosteroids**

Corticosteroids, known commonly as steroids, are used when patients cannot tolerate other anti-inflammatory drugs or they prove ineffective for an attack of gout. They are becoming popular in elderly people. Corticotropin (ACTH), a drug that converts to a steroid, is another option. (In one 2001 study corticotropin was more rapidly effective and had fewer side effects than indomethacin, the standard NSAID used for gout.)

Corticosteroids can be administered in different ways:

- If only one joint is affected, an injection of the steroid triamcinolone directly into the affected joint can often bring rapid pain relief.
- A single muscular injection of ACTH or triamcinolone may be the most rapid and reliable method for terminating an attack. Oral doses of prednisone are usually given for seven to 10 days after the injection in tapered doses. This is to prevent a rebound attack, which can occur after the injection.

These drugs should only be administered for short periods and not used for long-term treatment.

### **Uricosuric Drugs**

The uricosurics prevent the kidney from reabsorbing uric acid and so increase the amount excreted in the urine. They are usually the choice for preventing gout in the following patients:

- Those under 60 years old.
- Those with normal diets.
- Those who have normal kidney function.
- Those who have no risk of kidney stones.
- Uricosuric drug candidates should produce no more than 700 to 800 mg of uric acid in urine over a 24-hour period.

*Specific Uricosurics.* Probenecid (Benemid, Parbenem, Probalan) and sulfipyrazone (Anturane) are the standard uricosurics. An investigative uricosuric, benzbromarone, may prove to be beneficial, even in patients with some renal insufficiency.

Probenecid is taken two to three times a day and sulfipyrazone begins at twice a day and increases to three or four times daily. The initial doses should be low and then gradually built up. Probenecid combined with colchicine is more effective than probenecid alone, but patients respond differently to this regimen depending on the dosage balance, so it needs to be carefully individualized. A uricosuric combined with allopurinol [ *see below* ] is occasionally effective in cases where using just one drug is not.

*Side Effects.* The possible side effects of these two drugs include skin rashes, gastrointestinal problems, anemia, and kidney stone formation. To help reduce acidity and the risk for kidney stones, patients should drink plenty of fluids (ideally water, not caffeinated beverages). Sodium bicarbonate supplemented by acetazolamide can also reduce acidity and the risk for stones.

*Interactions.* Adding low-dose colchicine or an NSAID may help prevent gout attacks, but NSAIDs, particularly aspirin, as well as other salicylate drugs, interfere with uricosuric drugs and reduce effectiveness, so they should be avoided if possible. Patients who require minor pain relief should take acetaminophen (Tylenol and others) instead. Uricosurics interact with many other drugs, and a patient should be sure to inform the physician of any medications they are taking.

### **Allopurinol**

Allopurinol (Lopurin, Zyloprim) blocks uric acid production and is the drug most often used in long-term treatment for older patients and over-producers of uric acid (levels of

excreted uric acid are over 800 mg during a 14-hour period). It is also considered the drug of choice for patients with impaired kidney function, a history of kidney stones, and for tophaceous gout. (Preliminary research also suggests it may be helpful for patients with congestive heart failure, which has been associated with gout in some cases.)

*Administration.* Allopurinol is taken orally once a day in doses of 100 mg to 600 mg, depending on the patient's response to treatment. When it is first used, allopurinol can trigger further attacks of gout, and thus during the first months (or longer) of therapy the patient is also given a NSAID or colchicine to forestall that possibility.

*Side Effects.* Between 3% to 5% of patients experience severe side effects, diarrhea, headache, and fever. Among the more serious are blood cell abnormalities, including leukopenia (a reduction in the number of white blood cells) and thrombocytopenia (a reduction in the number of platelets). The drug may also increase the risk for cataracts. About 2% of patients experience an allergic reaction to allopurinol that causes a rash. In rare cases, the rash can become severe and widespread enough to be life threatening. Allergic individuals who had experienced only a mild rash may be able to build up their tolerance for the drug by undergoing a desensitization process.

*Interactions.* Allopurinol interacts with certain other drugs, such as azathioprine.

#### **Other Agents**

*Hypertensive Agents.* People with gout have a higher risk for high blood pressure. And some of the agents used for hypertension can increase the risk for gout attacks. Newer agents, such as losartan (known as an angiotensin II receptor antagonist), may have beneficial effects on both high blood pressure and gout.

*Urate Oxidase.* Recombinant urate oxidase (raburicase) is an agent being investigated to prevent hyperuricemia and gout in patients undergoing chemotherapy. It is proving to dramatically reduce uric acid levels and to be safe even in children.

#### **Alternative Agents.**

Some people use so-called natural remedies for gout. Patients should be very cautious when using such agents and do so only after checking with their physicians. [ *See Warnings on Alternative and So-Called Natural Remedies.* ]

#### **Warnings on Alternative and So-Called Natural Remedies**

It should be strongly noted that alternative or natural remedies are not regulated and their quality is not publicly controlled. In addition, any substance that can affect the body's chemistry can, like any drug, produce side effects that may be harmful. Even if studies report positive benefits from herbal remedies, the compounds used in such studies are, in most cases, not what are being marketed to the public.

There have been a number of reported cases of serious and even lethal side effects from herbal products. In addition, some so-called natural remedies were found to contain standard prescription medication. Of specific concern are studies suggesting that up to 30% of herbal

patent remedies imported from China having been laced with potent pharmaceuticals such as phenacetin and steroids. Most problems reported occur in herbal remedies imported from Asia, with one study reporting a significant percentage of such remedies containing toxic metals.

The following warnings are of particular importance for people with inflammatory disorders:

*Comfrey.* Comfrey is a herbal remedy commonly used for a number of inflammatory problems. There is recent evidence that comfrey can be toxic to the liver and animal studies have reported a possible cancer risk. It is banned in Canada and other countries but is widely available in the US.

*Gingko.* Although the risks for gingko appear to be low, there is an increased risk for bleeding at high doses and interaction with anti-clotting medications. Commercial gingko preparations have also been reported to contain colchicine, which is also used in gout. Patients should be aware of this possible ingredient.

The following website is building a database of natural remedy brands that it tests and rates. Not all are available yet. <http://www.ConsumerLab.com/>

The Food and Drug Administration has a program called MEDWATCH for people to report adverse reactions to untested substances, such as herbal remedies and vitamins (call 800-332-1088).

## WHAT LIFESTYLE MEASURES CAN HELP PREVENT GOUT?

### Avoiding Excessive Energy Demands

Any activities that increase energy demands also increase metabolism or purines that produce uric acid. Avoiding stress and staying healthy are important for preventing attacks.

### Dietary Recommendations

The American Medical Association recommends the following dietary balances for patients with gout:

- High in complex carbohydrates (fiber-rich whole grains, fruits, and vegetables).
- Low in protein (15% of calories and sources should be soy, lean meats, or poultry).
- No more than 30% of calories in fat (with only 10% animal fats).
- interest, however, was a 2001 study reporting a 67% reduction in gout attacks when patients limited their diet to 1,600 calories a day but only 40% were from complex carbohydrates while 30% were from protein and 30% from vegetable-based fats. Cholesterol levels also improved. More research is warranted on the optimal diet for gout patients.

*Reduce Foods Containing Purines.* Because uric acid levels are only minimally affected by diet, dietary therapy does not play a large role in the prevention of gout in the first place. Still, people who have suffered an attack of gout may benefit from reducing their intake of purine-rich foods if they habitually eat unusually large quantities of such foods. (Because purines are found in all

protein foods, no one should eliminate all purines.)

Purine-containing foods include the following:

- Beer and other alcoholic beverages.
- Anchovies, sardines (in oil), fish roes, herring.
- Yeast.
- Organ meats (eg, liver, kidneys, sweetbreads).
- Legumes (eg, dried beans, peas).
- Meat extracts, consommé, gravies. ( *Note:* Any meat, fish, or poultry has moderate amounts of purines. And diets high in protein, particularly animal protein increase uric acid. No studies have determined the value of reducing protein in gout patients, however.)
- Mushrooms, spinach, asparagus, and cauliflower.

*Possibly Helpful Foods.* Some specific foods may have benefits:

- Dark berries, such as blueberries, blackberries, and cherries, may contain chemicals that lower uric acid and reduce inflammation.
- Soybeans are also legumes, but one study of gout patients suggested that eating tofu, which is made from soybeans and is a source of complete protein, may be a better choice than meats.
- Certain fatty acids found in certain fish (eg, salmon), flax or olive oil, or nuts may have some anti-inflammatory benefits.

*Supplements.* Vitamin C and folic acid supplements may also have some benefits. Vitamin A, however, may *increase* the risk for gout attacks.

### **Maintain Healthy Weight**

A supervised weight-loss program may be a very effective way to reduce uric acid levels if the patient is overweight. Crash dieting, on the other hand, is counterproductive because it can increase uric acid levels and can cause an acute attack.

### **Maintain Fluids**

Drinking plenty of water and other nonalcoholic beverages helps remove MSU crystals from the body. Some researchers are studying the anti-inflammatory properties of green tea, which might have some benefit for gout. It should be noted, a Japanese study reported a higher association between gout and tea drinking (although the study did not describe the type of tea).

### **Avoid Alcohol**

Alcohol should be avoided, since it promotes purine metabolism and uric acid production; it also may reduce excretion of uric acid. Heavy drinking, especially binge drinking of beer or distilled spirits, should especially be avoided.

### **Avoid Joint Injury**

People with gout should also attempt to identify and avoid activities that cause repetitive joint trauma, such as wearing tight shoes.

### **Preventing an Attack During Travel**

Travel is an example of an activity that increases the risk for gout. It not only increases stress, but eating and drinking patterns may change. Before traveling, patients should discuss preventive measures with their physicians. The doctor may prescribe a prednisone tablet to be taken immediately at the first sign of a gout attack; in most cases this stops the episode.

## **WHERE ELSE CAN HELP BE OBTAINED FOR GOUT?**

The Arthritis Foundation, 1330 Peachtree St., Atlanta GA 30309. Call (800- 283-7800) or (<http://www.arthritis.org> ).

An excellent source for many types of services. The foundation funds research and provide brochures, video tapes, exercise programs, physician referrals, and local chapters.

Takeda Pharmaceuticals North America, Inc. <http://www.gout.com>

The National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS). One AMS Circle, Bethesda MD 20892-3675. Call (301-495-4484) or (<http://www.nih.gov/niams> ).

American Academy of Orthopedic Surgeons. PO Box 2058, Des Plaines, IL 60017. To receive information on arthritis, send a self-addressed stamped business envelope. Be sure to designate "Arthritis" on the mailing envelope.

Aids for Arthritis, Inc. Call (609- 654-6918).  
Offers supportive devices and products for people with arthritis.

Enrichments. Call (800- 323-5547) or (<http://www.sammonspreston.com> ).  
Offers supportive devices and products for people with arthritis.

